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ABSTRACT

A scheme of classification is suggested for physicians faced with various prescriptions for remediation of children with learning disabilities. Three models of the causes of learning disability are suggested: (1) the difference model which stresses normal variability in the pattern of development of mental abilities; (2) the deficit model which assumes a limited malfunction of the brain; and (3) the delay model which suggests transitory immaturity or lag in development as the cause for learning disability. Process-oriented and goal-oriented strategies of management are delineated. Remedial methods are classified according to their implicit model of etiology and their adherence to the process- or goal-oriented approach. An atheoretical, goal-oriented approach which works with the existing pattern of the child's abilities and seeks to tailor individualized instruction to the specific gap in academic understanding and knowledge is advocated. (Author/ED)

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MODELS OF LEARNING DISABILITY: THEIR RELEVANCE TO REMEDIATION

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Children with learning disability are those who, though normally intelligent and motivated, experience an unexpected degree of difficulty in mastering some aspect of the customary school curriculum. Faced with such children, clinicians have responded with a bewildering miscellany of remedial recommendations. It helps one to understand and evaluate these recommendations if one realizes that each is based on some particular, implicit assumption about the circumstances that underlie the behavioural disability. We will make explicit three distinct models of learning disability as well as two quite different approaches to resolving the problems that the dsiability creates. These are the difference, deficit and delay models, and the process-oriented and goal-oriented strategies of management.

A process-oriented strategy of management basically follows the medical model. This consists of determining the surface manifestations, inferring an underlying abnormality, and instituting therapy intended to restore that underlying abnormality to normal. It should then follow that the abnormal symptoms and signs with which the patient presented will resolve, leavning no further cause for complaint. In the area of learning disability, a process-oriented approach would seek to determine the particular abnormality of brain function that underlies the behavioural difficutly and to attempt to restore the brain processes to normal. It would then be supposed that the child's behaviour would normalize and the usual social goals would become possible for the child.

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The goal-oriented approach recognizes the lack of evidence that brain-based processes can be modified with any means at present available. Whether modification of brain-based processes will become possible in the future is a question that is not relevant to action now on behalf of a child. The goal-oriented approach designates an academic objective; it's intended to reconcile necessities imposed by the social setting with the highest potential that the child could realize under existing circumstances. The view is taken that any deficit is compounded by underachievement relative to that deficit. A child who cannot learn in a regular setting but who could learn at a slower rate, with teaching geared to his individual learning requirements, will fail even to learn at that slower rate unless specific arrangements are made. This deficit-related underachievement is based on a series of failures and decreased motivation, due to instruction not geared to the child's individual need. It is so pervasive that one could attribute children's gains in remedial programmes to the fact that these programmes provide individualized instruction. Individualized instruction allows the teacher to teach at the child's level rather than, as in a large class, assigning work the child cannot possibly do. In the latter case, the child learns practically nothing and thus underachieves even for himself, failing to learn what he could learn if it were offered. With individualized instruction the child learns as much as he is capable of learning, and so he neither underachieves (relative to himself) nor experiences repeated failure. Those factors probably form the core of reasons that children improve in remedial programmes, rather than that, the child's mental processes and learning potential have been affected. The simple and primary method is to teach children at their appropriate level. This is the pragmatic approach, which is based on existing reality and directly aimed at better equipping the child with academically and socially relevant skills, rather than hoping indirectly to affecting his learning potential by acting on the brain. Thus, the models that will now be discussed do not help decide how to help a failing child. Their value is heuristic.

The difference models stresses normal variability in the pattern of development of mental abilities. The deficit model assumes a limited malfunction of brain. The delay model deals in terms of transitory immaturity or lag in development.

Individual differences are no less to be expected in cognitive than in physical development. They involve not only the child's overall intelligence but also the degree of excellence he achieves with respect to the various components of that intelligence. Just like adults, some children will show more aptitude for verbal, others for spatial tasks. Some will be more systematic, others more creative. The diverse opportunities offered by adult employment provide scope and application for many different patterns of ability. The school system, however, is restrictive and traditionally rigid in the demands it makes on the pupil's intellect. Those children whose relative weakness is in verbally-related skills, such as reading, will be labeled "weak students," regardless of how talented they may be in other respects. The atmosphere of failure can easily be a self-fulfilling prophecy, and as the child's motivation drops, the arena of failure enlarges to encompass the whole of his school performance.

Few would deny that there is a genetic omponent in individual differences in intelligence. But, in addition, children model themselves on their parents, whose attitudes and interests may be restricted and widely divergent from those valued in the typical school. Some believe that such cultural differences result in deprivation with respect to certain kinds of experience and that this deprivation in turn results in an underdevelopment of relevant brain processes. If depriving a child of certain experiences results in selective brain underdevelopment, then it can be easily supposed that supplying those experiences would correct the unevenness of brain development. This stimulation or enrichment therapy is different from instruction, because it does not so much seek to acquaint the child with specific items of information that he should know;

rather, it tries to supply him with an environment intended to stimulate his brain processes to develop the capability readily to absorb the information in question. The difference model is applicable not only to styles of thinking but also to styles of attending. Just as enrichment is intended to supply a verbal and problem-solving approach, so it is intended to inculcate reflective and conformist ways of attending. The hyperactive, distractible, and impulsive child might be regarded as having lacked in his home environment the necessary structure and model of organized attending to develop his own capability in these respects. Enrichment therapy would then logically supply the kind of structured environment that had been lacking.

We see, then, that the difference model deals in variability of brain development but focuses its emphasis on the interaction between early experience and an adaptively relevant repertoire of intellectual skills. The deficit and delay models are, in contrast, posited on organic origins for the resultant behavioural patterns.

The deficit model, a traditional medical model, generates a quite different perspective and leads to different recommendations. The learning disabled child has a deficit in his area of weakness; it may be language, reading, arithmetic, or something else. An underlying brain lesion is proposed which may or may not be revealed by history taking, physical examination, and the usual neurological investigative programme. The concept of the "brain-damaged child" will lead to two quite different sets of recommendations, depending on the clinician's attitude towards the mutability of a damaged brain. Those who regard brain damage as in principle irrevocable will recommend the facing of "reality" on the part of the parents and child, with consequent adjustment of expectations and an educational programme geared to the situation as it exists, incorporating a pessimistic prognosis. Whereas on a difference model the affected child would be seen as developing in parallel to the norm though a constant amount below it, the deficit model would see a widening gap as normal brain further develops but abnormal brain does not do so.

Others might adhere to this model, but believe that damaged brain can be helped or made to function normally by being stimulated. A further refinement might propose that whereas stimulation in the "affected modality" might not be effective, "flooding" the system with polysensory stimulation somehow restores damaged brain to normal function. Here again we have a programme which is not geared to educational needs but rather floods the child with input in the hope that this will change the central organization. It is as though by making more telephone calls one could change the structure and organization of the central telephone exchange. Specifically, the messages are credited not only with the function of transmitting information but also with the function of stimulating the central connections that they reach.

The delay model stresses the rate of acquisition of abilities. The learning disordered child is selectively immature or unready. With respect to the affected function he is like a normal younger child. The learning disordered children are not qualitavtively affected or transformed by "brain damage" into a different functioning organism. They are different only with respect to the age at which they attain certain capabilities, not in the manner in which those are at long last attained. The "soft" neurological signs of minimal brain dysfunction are a case in point. These manifestations are not grossly abnormal at any age, in contrast to, for instance, a hemiplegia or a visual field defect. Rather, they represent a relatively long persistence of primitive behaviour patterns and the delayed acquisition of more sophisticated forms of control; in effect they mostly relate to the persistence of synergisms and associated movements, characteristic of the "clumsy" child. Consider hyperactivity. Whereas a deficit model would view this as a pathological failure to control impulses, a delay model assumes delayed development of that control.

On this view the hyperactive child would be like a younger normal child in his relatively ineffective impulse control.

The delay model does not seek to explore pathogenesis in any detail. Whereas those who think in terms of deficits might well ask which part of the brain is damaged, and look for structural change either at the macroscopic or microscopic level, delay theorists would minimize the expectation of histological changes (although they would not discount that possibility). After all, the effect of early focal brain damage on areas that have not yet assumed mature function is in any case to induce a delay in the assumption of that function. Only in the unusual case is that delay so extreme that for practical purposes no behavioural gains are made over time. However, plateaus in development would not necessarily indicate such underlying damage, as it is by no means unexpected for children to show stops and starts in development of any aspect of their bodily maturation. After all, some normal children walk at the age of 9 months and some not until the age of 18 months, but once they are walking, who can tell the difference?

Most important, the delay and deficit models raise different prognostic expectations. Whereas the outcome on a deficit basis is necessarily gloomy, the delay theory envisages a gap in ability that gradually closes over time. Thus, the deficiency in impulse control of the hyperactive is expected to resolve with maturation so that even if later than usual, ultimately the desired end-point of impulse control will be achieved, and the syndrome will have resolved. Whereas on a deficit basis one might expect the hyperactive children would retain their impaired impulse control into their adult years (where it might manifest in a manner characteristic of that stage of development by inducing psychopathic or characterological social pathology), the delay theorist might be more optimistic in his expectations for a normal adjustment in due course.

The advice given by adherents of the delay model will again depend on their notion as to whether the rate of cerebral maturation can be accelerated by physical measures. Those who think it cannot will counsel patients to concentrate on existing strengths and to avoid areas of weakness. They will advise that morale should be maintained pending the arrival of cerebral maturation and, therefore, of the delayed behavioural capability. Others, however, will feel that lags in cerebral maturation can be corrected. For instance, a delay in language development might be attributed to a delay in the lateralization of language in the left cerebral hemisphere. On the basis of such an assumption some delay theorists try to manipulate hand preference and otherwise emphasize use of the right side of the body in action, supposing that this is some way would help distribute language function to the left side of the brain. But as with all other theorizing about the effects of external changes on human brain development, the evidence for this is lacking.

Thus, process-oriented clinicians will differ in their history-taking, etiological theorizing, prognostications and management, depending upon which model they adopt. In history-taking, the difference model focuses attention on the child's early environment, the deficit model leads one to question the possibility of pathological events before, during, or soon after birth, and the delay model would lead one to look for comparable, presumptively genetic events in the family history.

In explaining the situation to parents and others the difference theorist will talk in terms of environment, the deficit theorist in terms of brain damage, and the delay theorist in terms of brain immaturity. Difference theorists will explain that the child will develop in parallel to, though at a lower level than, unaffected children. Deficit theorists will be gloomy about prospects for development in the area of difficulty, whereas delay theorists will regard that development only as a matter of time.

As regards management the difference emphasis will be on manipulating the environment, the deficit emphasis might be on sensory stimulation, and the delay emphasis on physical manipulations intended to hasten the settling down of particular processors into their "mature" form of cerebral organization.

In evaluating various positions, one should not be misled into seeking to prove them incorrect. The onus is on the proponents of a particular model to supply adequate evidence for its acceptance. While that evidence in medicine need not be watertight before it can be acted upon, there should be both a suggestive clinical experience and some rational theoretical basis before one commits the physical, financial, and emotional energies of one's patients to often arduous programmes which, if ineffective, will only deepen the sense of failure and frustration within the family group. Pending such reasonable evidence, we would advocate an atheoretical, goal-oriented approach which, while leaving under constant review the possibility of change in the pattern of a child's abilities, works with the existing pattern and seeks to tailor individualized instruction to the specific gap in academic understanding and knowledge. Basically, this approach goes back to the earliest discernible academic difficulty. What was the last thing the child understood and the most difficult thing that he has mastered? At what point did he fail to understand what other children did understand and, therefore, make inadequate further gains? A goal-oriented approach is conservative as regards prognosis, pragmatic as regards the means which it adapts to the goal in view. In contrast to the process-oriented attempt to change the child's brain to make him capable of benefiting from the customary instruction, the goal-oriented approach adapts the learning disabled child.